Some Hitherto Undescribed Symptoms in Angina Pectoris

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A Clinical Lecture delivered in the Royal Infirmary

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Some Hitherto Undescribed Symptoms in Angina Pectoris.

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Although angina pectoris was the subject of one of my clinical lectures last term, the subject is so important as to merit further study. We have, moreover, at present in our wards the most interesting example of the affection which it has ever been my good fortune to observe. The complex of symptoms presented by this patient is so extraordinary that it seems my duty to lay the facts before you in full detail. They will furnish an occasion for amplifying the remarks previously made on the modern conception of the condition.

A labouring man, aged 45, born in Leitrim, but resident for some years in Edinburgh, was admitted to my ward on account of pain in the chest and left arm. His father lived to the age of 85, and was said to be subject to rheumatic pains. His mother died, when 33 years old, of puerperal causes. The patient says he was never addicted to alcoholic excess, seeing that for him it was too expensive a luxury. He has been in the habit of smoking about two ounces of strong tobacco every week. His home conditions are healthy and comfortable, but his work is rather severe. When 21 years old he suffered from some fever, as to the nature of which he is ignorant. In more recent years he had an attack of pneumonia. The commencement of the present illness appears to have been about six weeks ago, and it seems to have begun with breathlessness on exertion; the actual onset of the pain in the chest and arm was five weeks before admission. Its first appearance was when he was in a train on the way to his work. He was able to perform his

duties for three days, but since then has been unfit for any exertion. The pain has persisted continuously ever since its first onset, but two weeks after it began it underwent a change. At first it was confined to the region of the left scapula, but it afterwards began to shoot down the left arm, and at the same time it showed paroxysmal exacerbations.

The present condition of the patient is as follows :—He is a strongly-built man, 5 ft. 7 in. in height, weighing 10 st. 1 lb., with a healthy, florid complexion.

The circulatory system gives evidence of considerable alteration. The patient, as a rule, has a high complexion, except during a paroxysm of pain, when he becomes paler. There is some dilatation of the venules on the cheeks and nose, and the temporal arteries stand out in a sinuous fashion, although there is no excessive pulsation in them or in the carotids. The superficial arteries are thick and tortuous. The arterial pressure is high at all times; measured by Hill and Barnard's sphygmometer it varies between 160 and 170 mm. Hg. During the paroxysms of pain the pressure

Fig. 1.
Usual pulse. Pressure 4 oz.

rises considerably, but for obvious reasons it is then difficult to obtain an exact measurement. The pulsation is perfectly regular and not unduly frequent; each individual pulsation

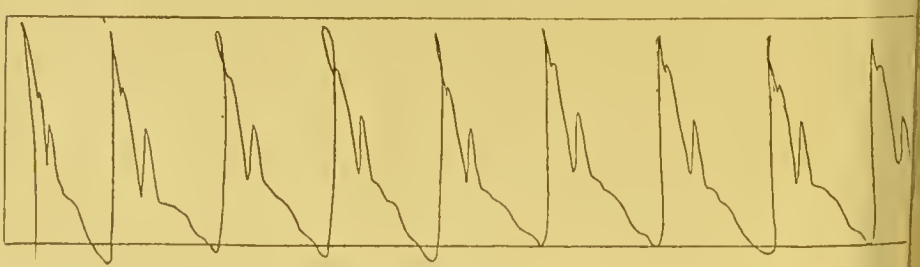

Fig. 2.
Usual pulse. Pressure 5 oz.

is of large amplitude and is well sustained. Tracings of the ordinary pulse are given in figs. 1 and 2.

The Junior House Physician was in the act of taking some sphygmographic tracings when one of the paroxysms of pain set in, and he found that with the increase of pressure there was a characteristic reduction in the size of the pulse, with some acceleration, as shown in fig. 3, while,

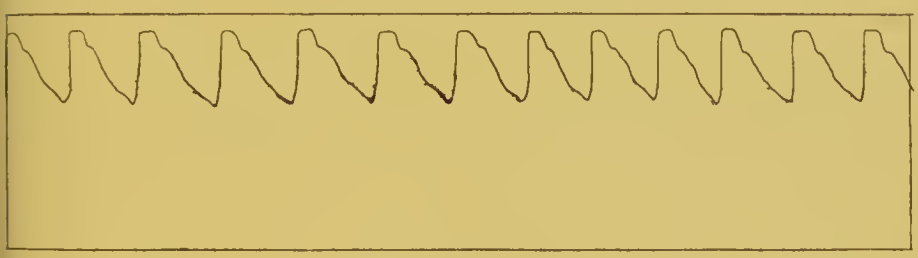

FIG. 3.
Pulse during paroxysm of pain. Pressure 3 oz.

after the use of nitrite of amyl and the relief of pain, the pulse became large, bounding, and still more frequent, as

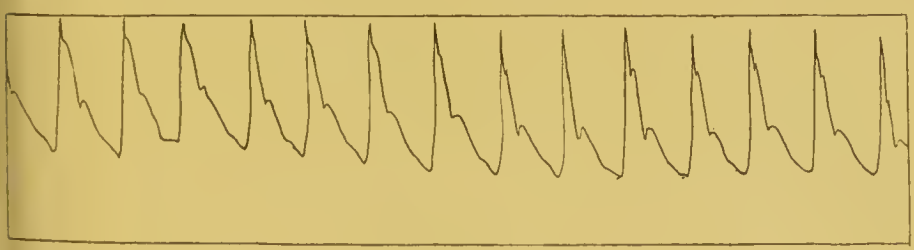

FIG. 4.
Pulse after nitrite of amyl. Pressure 3 oz.

in fig. 4. The case, in respect of the pulse, therefore, conforms to the type of angina with spasm of the arterioles.

On inspection, the apex beat is seen in the fifth intercostal space, about the mammillary line. There is no other impulse visible in any part of the præcordia or epigastrium. On palpation, the apex beat is found to be situated $4\frac{1}{2}$ in. from mid sternum. The impulse is forcible and without any thrill. The superior border of the heart on percussion is found at the upper edge of the third left costal cartilage.

The left border is $4\frac{3}{4}$ in. and the right is 2 in. from the middle line. The heart sounds are perfectly clear, but the aortic second sound is much accentuated.

The most careful investigation of the chest fails to give any evidence of other disease, and the X-rays, employed both by screen and by skiagraphy, show a perfectly normal state of matters at the base of the heart.

The patient complains of an extensive and constant pain, most severe, as a rule, over the back, where it extends down as far as the ninth intercostal space. It reaches up to the back of the neck, and passes round its side so as to reach the left clavicle, which it crosses, and extends down the middle

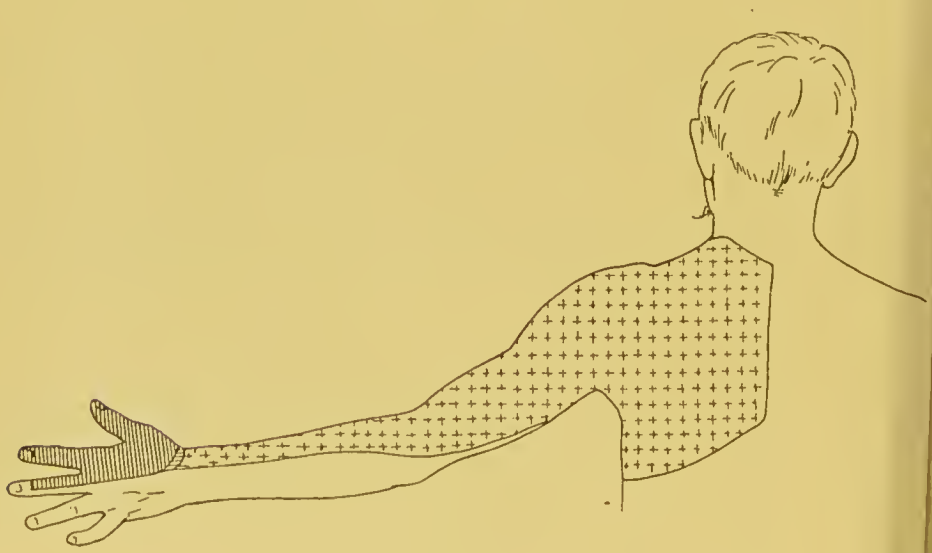

FIG. 5.

Distribution of sensory changes posteriorly. The crosses denote hyperæsthesia; the horizontal shading analgesia without anæsthesia; the vertical shading analgesia with anæsthesia.

line to the eighth intercostal space, from which point it becomes continuous with the pain at the back by passing round the inferior axillary region. From the shoulder it extends down the outer aspect of the arm and forearm so as to reach the thumb, index and middle fingers. The exact boundaries of the pain can be seen in the figs. 5 and 6, drawn from photographs taken after the areas had been marked on the skin. When the paroxysms of pain occur, the boundaries appear to the patient to be somewhat enlarged, and it

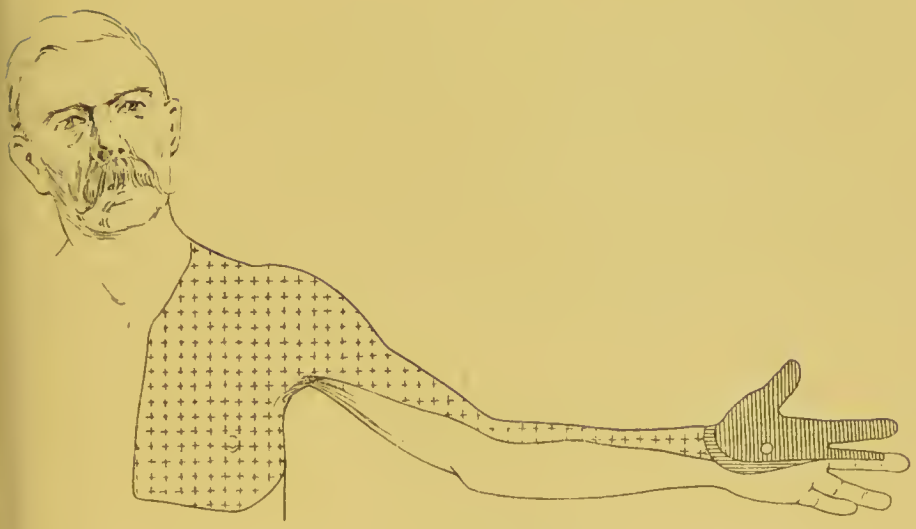

FIG. 6.

Distribution of sensory changes anteriorly. Explanation as in fig. 5.

shoots up the left side of the head so as to involve the cranial vault as far as the middle line, and includes most of the arm and forearm.

On testing the sensibility of the surface, a most interesting group of phenomena can be discovered. Over the whole

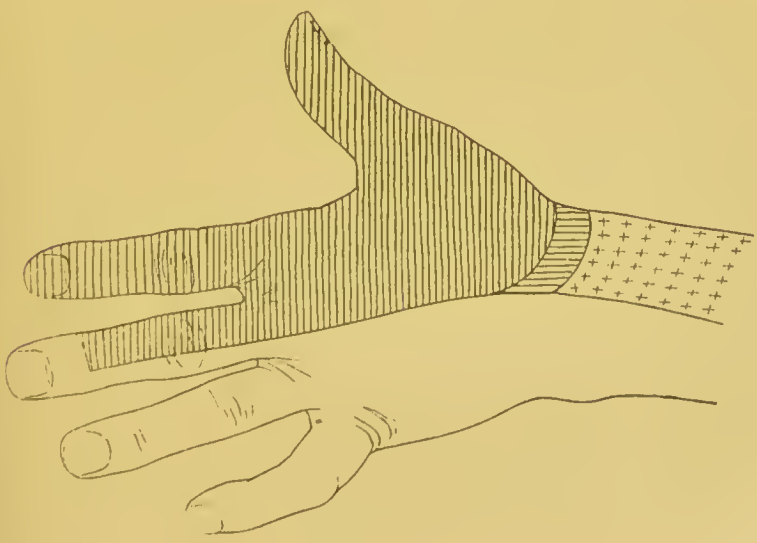

FIG. 7.

Sensory changes in back of hand. Explanation as on fig. 5.

of the area marked with crosses there is a high degree of hyperæsthesia to every form of stimulus, but over the radial portion of the hand there is absolute anæsthesia, bounded along part of its edge by a small zone of analgesia. The anæsthesia is complete to every kind of stimulus, even to the most intense we can employ ; the analgesic area is quite sensitive to all ordinary tests, but it is impossible to elicit the least sensation of pain. The exact distribution of this is marked upon the patient by horizontal and vertical

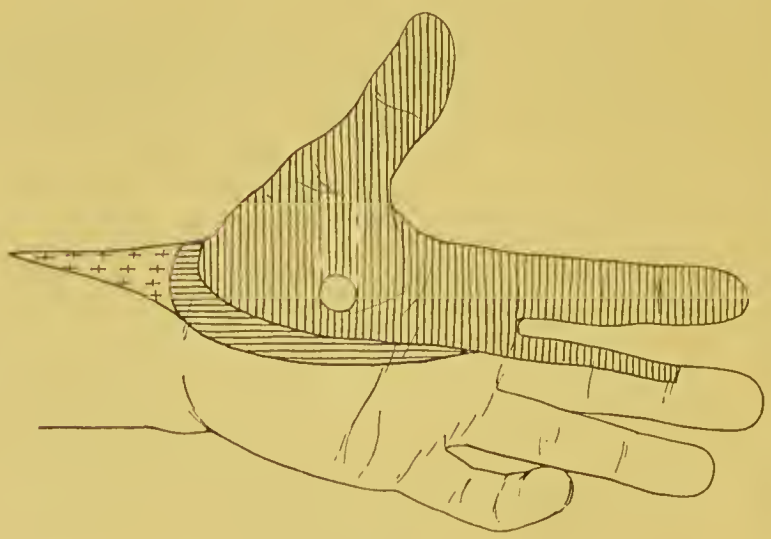

FIG. 8.

Sensory changes in front of hand. Explanation as on fig. 5.

lines, and it may be seen in figs. 7 and 8. The anæsthesia is profound to every form of stimulus, but there is one small point about the size of a three-penny piece at the junction of the thenar eminence and the palm of the hand in which the patient feels perfectly well. Probably, as Professor Cunningham has suggested to me, this may be due to a communicating branch from the ulnar nerve.

Another point of interest is that the sterno-mastoid, trapezius, pectoralis, deltoid, biceps and supinator muscles of the left arm are much more tender than those of the right. There is no other sensory disturbance and the special senses are intact.

Now, before leaving the afferent nervous system, it is necessary to point out two divergences from the usual state of matters exhibited by our patient. One of these may be due to an anatomical peculiarity. It is the line of division separating the insensitive from the sensitive part of the palm. Instead of being bounded by the middle of the ring finger, as is usual in front, the line of demarcation of ulnar and median nerves runs along the middle of the middle finger, unless we may assume that a portion of the median nerve has escaped. The other departure from what is customary is much more difficult to understand. As will be observed from the outlines painted on the patient, the inner or ulnar aspect of the arm, forearm, and hand, is not affected, *i.e.*, a considerable area belonging to the segmental distribution of the seventh and eighth cervical and first dorsal portions of the cord is intact, while the areas above and below are deeply involved. Now the segments unaffected in our case are those usually most deeply involved. In view of the cilio-spinal effects about to be described, this fact is difficult to understand, and it seems to me to indicate a capriciousness of impression on the part of the spinal segments.

The organic reflexes are normal. The cutaneous reflexes are all present and appear to be within usual limits. The supinator and triceps jerks are increased in the left arm as compared with the right, and the myotatic irritability is increased in the deltoid and the other muscles on the outer aspect of the left arm and forearm. There is great weakness of the left hand, especially in the thumb and first and second fingers, so that the patient's grasp is practically restricted to the ring and little fingers. With the dynamometer the grasp of the right hand is 135 and of the left 50. There is wasting and loss of tone in all the muscles of the shoulder-girdle, arm, forearm, and hand on the left side. The right arm is in circumference 12 in., the left 10½ in. The right forearm is 10½ in., and the left 10 in. On testing the electric reactions an increased excitability is found in the muscles which are affected. The exact state of matters will be easily understood from the following table, in which the

measurements of faradism are expressed in the number of centimetres on the scale of the induction apparatus, while those of galvanism are given in milliampères :—

Faradism.

	R.	L.
Deltoid	2.7	2
Biceps	1·6	1·4
Supinator longus	1·9	1·2
Flexor sublimis	3·2	1·9

Galvanism.

	R.	L.
Deltoid	9 ccc. 10 acc.	6 ccc. 7 acc.
Biceps	5 ccc. 6 acc.	3 ccc. 4 acc.
Supinator longus ...	6 ccc. 7 acc.	5 ccc. 6 acc.
Flexor sublimis... ...	11 ccc. 12 acc.	9 ccc 10 acc.

One other point deserves consideration. The left eye is more prominent than the right, and the left pupil is larger than its fellow. This condition has been present throughout his residence in the ward, but it is more marked at those times when the pain gives him serious trouble. There cannot be a doubt, therefore, that there is irritation of the sympathetic nerve on the left side, involving the cilio-spinal fibres. It is interesting to observe that these phenomena are not accompanied by any alteration in the functions of the skin. There is no redness or increase of perspiration, but, as already mentioned, there is some general pallor of the whole countenance during an attack.

The diagnosis in this interesting case is early arterial sclerosis, probably implicating the coronary arteries, and accompanied by some interstitial changes in the myocardium —the combination, in short, which is so commonly attended by angina pectoris. But, as already remarked, there is a vaso-motor element in the attacks ; this, as is universally recognised, is a symptom frequently met with.

Numbness of the arm, the forearm, or the hand, has often been observed in cases of angina pectoris, but complete anæsthesia, such as is present in this case, must be rare indeed; in fact, no instance even distantly approaching that just described, has ever come under my notice. Neither in the beautiful researches of Mackenzie,[1] nor of Head,[2] is there any reference to such conditions. In a private communication, however, the former states that he seemed to obtain anæsthetic areas when he first investigated the sensibility in angina pectoris, but that in late years he had failed to find them and concluded he had made a mistake.

The wasting of the muscles has been described both by Eichhorst[3] and myself,[4] and there is no difficulty in explaining such trophic results from powerful afferent impulses. They are strictly analogous to the conditions which are found in arthritic muscular atrophy. This has long been considered as due entirely to reflex influence, which has been fully discussed by Paget[5] and by Vulpian,[6] and the theory is strongly supported by the fact that in experimental joint lesions wasting of the muscles is prevented by previous division of the posterior spinal roots, which has been described by Raymond.[7] But there are some distinct differences between the wasting in this case and in that of arthritic muscular atrophy. In this patient, just as occurs in arthritic atrophy, there is considerable increase in myotatic irritability. But no case of arthritic dystrophy of muscles has ever been under my notice in which there was an increase in the electric irritability of the atrophied muscles. It is possible, however, that what we are dealing with is an early condition, and that a reduction in electric irritability may be a later phase.

In addition to providing a number of hitherto undescribed symptoms for our study, the patient seems to me of the deepest interest as giving proof of a means of communica-

[1] *Medical Chronicle*, 1892, p. 293; and *Lancet*, 1895, Vol. I., p. 16.
[2] Brain, 1893, vol. xvi., p. 1, and 1894, vol. xvii., p. 339.
[3] "Handbuch der Specieller Pathologie und Therapie," 1895, S. 231.
[4] "The Nervous Affections of the Heart," 1904, p. 18.
[5] The *Lancet*, 1873, Vol. II., p. 727.
[6] "Leçons sur l'Appareil Vaso-moteur," 1875, Tome 2, p. 327.
[7] *Revue de Médecine*, 1890, p. 374.

tion between the heart and the nerve centres which has hitherto received but scant attention.

While the vagus centripetal mechanism has been long recognised, that of the sympathetic system has been a matter of great doubt. In my Morison lectures,[1] the course of these tracts was fully described and the conclusions then reached may be now briefly summarised. Certain channels, proved by undoubted clinical facts to supply the course of afferent impulses, have not yet received support from any experimental evidence. The afferent impulses passing upwards in connection with the inhibiting and dilating mechanism are carried by the vagus nerve. The afferent impulses belonging to the accelerating and constricting mechanism must travel by the inferior cardiac nerve to the inferior cervical ganglion and thence by the gray rami to the seventh and eighth segments of the cord. It is possible that they may also pass round the annulus of Vieussens to reach the upper thoracic segments. But of afferent nerves belonging to the heart and passing to the spinal cord, there is no experimental proof. If we may judge afferent nerves by those of the visceral system of the abdomen, they are always medullated up to the posterior ganglion, and many of these medullated fibres are of large size. Reasoning by analogy it might be expected that the afferent cardiac fibres must also be medullated, but Gaskell,[2] when working at the fibres of the dog's heart was much struck to find that all the nerves from the annulus of Vieussens to the heart were non-medullated—no medullated fibres were traced to the depressor or the vagus nerve. He therefore concluded that the anatomical evidence confirmed the physiological, and that both pressor and depressor fibres reached the central nervous system by the vagus nerve. By means of a private communication he has, however, expressed his opinion to me that the spinal heart nerves are only a special example of the system of vaso-constrictor nerves, and therefore, if the blood-vessels possess

[1] *Op. cit.*, p. 32.

[2] *Transactions of the International Medical Congress, London*, 1881, vol. i., p. 254; *Philosophical Transactions, London*, 1883, vol. clxxiii., p. 993; *Journal of Physiology*, 1884, vol. v., pp. 46 and 362; *Ibid.*, 1886, vol. vii., p. 1.

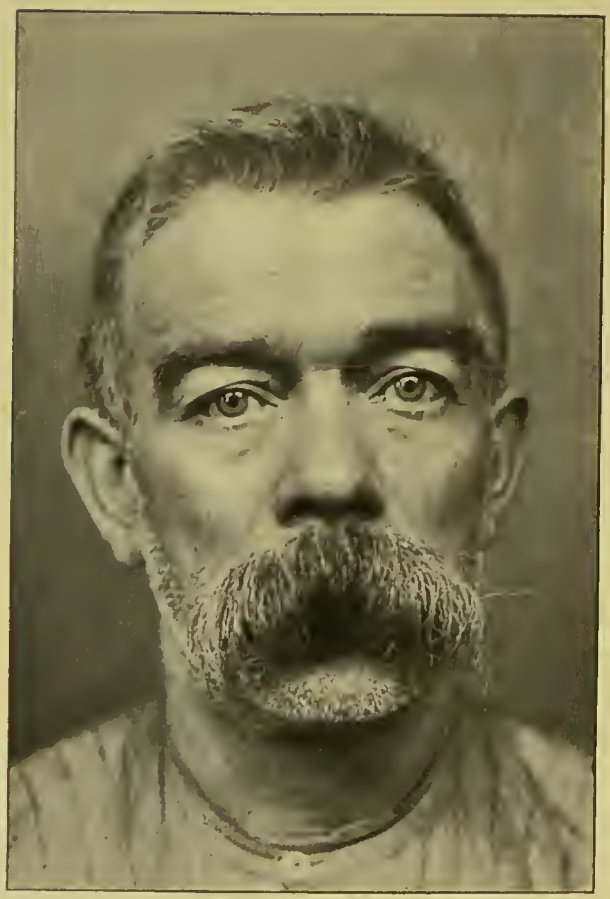

FIG. 9.

From a photograph of the patient, showing prominence of the left eye and dilatation of the left pupil.

afferent nerves, the heart, in virtue of its connection with that system must possess such nerves. He believes that there is evidence of the possession of spinal afferent nerves by the blood-vessels, while there is no evidence to prove that the blood-vessels possess any number of medullated fibres. His view is accordingly that it is possible, indeed probable, that afferent nerves, at least from the coronary vessels, should accompany the accelerator and so account for the clinical facts.

The real importance of this case, then, in my opinion, lies in the opportunity which it affords of giving a clear demonstration of certain facts regarding the afferent impulses from the heart; the symptoms exhibited by the patient allow the possibility of tracing out the course of these paths. In the diagram, fig. 9, the direction of these channels of communication is traced out. Passing up from the receptive end organ (*H*), by the cardiac nerves to the cervical ganglia (I *G*, II *G*, and III *G*), of the sympathetic, they pass in, as has been elsewhere shown by me,[1] by the gray rami communicantes (*PC*), to the posterior spinal roots, and thence run upwards in the ascending tracts of the spinal cord. In the cortex cerebri (*C*), the impulses which are produced give rise to sensation, but, as Head[2] puts it, "the sensory and localising power of the surface of the body is enormously in excess of that of the viscera, and thus by what might be called a psychical error of judgment, the diffusion area is accepted by consciousness and the pain is referred on to the surface of the body instead of on to the organ actually affected." The result is that the patient seems to feel uneasy or painful sensations over the præcordia, shoulder and upper extremity, as at *S*. That the cervical sympathetic is involved in this case cannot be denied on account of the ocular phenomena, and our patient may be taken as a proof of the contention that this system is the means of conducting afferent impulses from the heart. The figure (fig. 10) gives a diagrammatic representation of part of the cilio-spinal mechanism, showing the course of the spinal fibres

[1] *Op. cit.* p. 30.
[2] *Op. cit.*, vol. xvi., p. 126.

from the pupil-dilating centre (*PD*) in the base of the brain down to the cilio-spinal centre (*CS*), and thence by the anterior spinal root and anterior or white ramus communicans (*AC*), to the sympathetic system, whence the fibres pass upwards to the radiating fibres of the iris (*DP*) to the muscle of Müller (*MM*), and to the non-striped

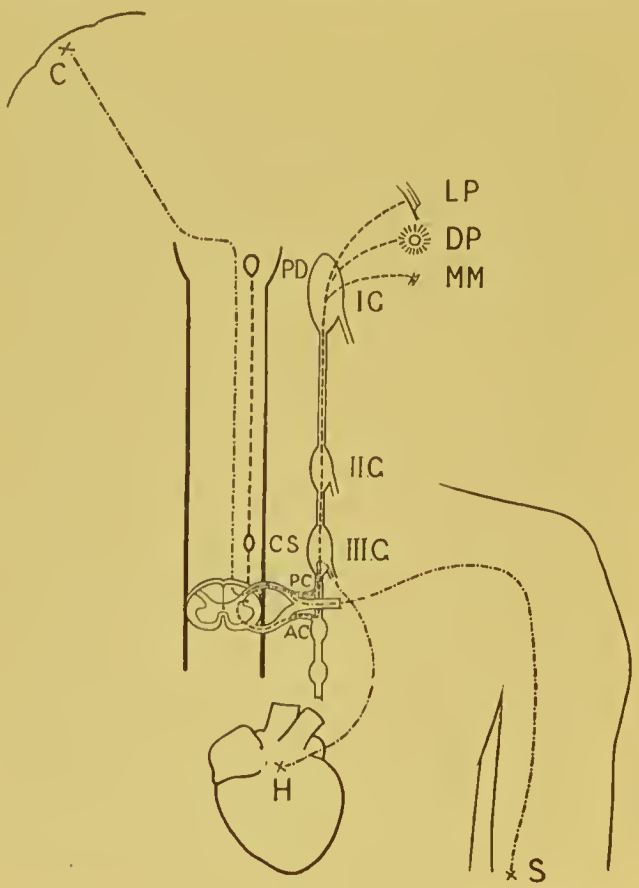

Fig. 10.

Course of impulses in the case. The dots and dashes indicate afferent—the dashes alone efferent tracts.

portion of the levator palpebræ (*LP*). In the construction of this diagram, a figure designed by Purves Stewart[1] has been very useful, and it is a pleasure to acknowledge its assistance. For untiring assistance in unravelling the details of this very interesting case my warm thanks are due to my House Physicians Dr. R. W. Johnstone and Dr. H. A. Stewart.

[1] *The Practitioner*, 1905, vol. lxxiv., p. 189.

Since the delivery of this lecture the patient has been almost continuously under observation, and the course of the affection must be mentioned. He was treated by iodide of potassium in ten grain doses three times a day, along with inhalations of amyl nitrite whenever a paroxysm of pain occurred. In the course of a week the severe attacks disappeared, and in less than a month the hyperæsthetic and anæsthetic phenomena were greatly diminished. But at the time of writing this additional note, four months after the patient came under treatment, there are still remains of the motor, electric and trophic changes, as well as some prominence of the left eye, and slight dilatation of the pupil. The hyperæsthesia is still slightly present, but in an almost inappreciable degree. Of the anæsthesia not a trace now remains.